Hazardous Child Labor: Lead and Neurocognitive Development

LISA S.R. IDE, MD, MPH[a]
DAVID L. PARKER, MD, MPH[b]

Hazardous child labor is challenging to define and quantify in the context of acute or chronic toxic exposures—either of which may cause significant disease and disability. Epidemiologic occupational studies in adults have documented many harmful outcomes secondary to exposure to toxic substances. Occupational surveillance efforts often have focused on acute injuries because they are more readily identified. Fassa has been able to compile data concerning injuries to child laborers but notes, "There is great need for studies in developing countries . . . on the impact of child labour on illness."[1] Although injuries may be underreported or undocumented among child laborers, acute injury is, at least, potentially recordable. This is in sharp contrast to toxic exposures, where exposure assessment is usually difficult and costly. This is especially true when it is done after an exposure has taken place.

The outcomes of most toxic workplace exposures to children remain unknown. The Agency for Toxic Substances and Disease Registries (ATSDR) notes that in similar environments, children may have greater exposures than adults: "Pound for pound of body weight, children drink more water, eat more food, and breathe more air than adults," and "In some instances, children are less able than adults to detoxify chemicals and are thus more vulnerable."[2]

Children who begin work at an early age have many more years to develop illness than an adult doing the same work. A household survey of child laborers in Ethiopia found that a high proportion (greater than 90%) of children in both urban and rural areas of the country reported non-use of protective equipment.[3] However, it is the experience of present author D.P. that this equipment is rarely if ever adequate, even when made available.[4] For example, protective equipment such as respirators or impermeable gloves are designed for adults, and thus do not properly fit children.

In defining hazardous child labor, the larger context of public health cannot be ignored. The study of child labor needs to take into account the baseline health of exposed individuals. In developing countries, the poorest and most vulnerable children are most often involved in work in order to earn money for survival. These children are also likely to already lack basic necessities of food and medical care, predisposing them to diarrhea, anemia, and micronutrient deficiencies. Underlying nutritional conditions may make children more susceptible to the effects of toxic substances such as lead.[5] In addition, child laborers also may be exposed to lead and other toxic substances from their poor living conditions.

[a]Employee Occupational Health Services, Fairview Health Services, Minneapolis, MN

[b]Park Nicollet Clinic, Minneapolis, MN

Address correspondence to: Lisa Ide, MD, MPH, Employee Occupational Health Services, Fairview Health Services, MB247A, 2450 Riverside Ave., Minneapolis, MN 55454; tel. 612-672-7469; fax 612-273-4723; e-mail <rage0001@umn.edu>.

Prenatal exposure to toxic substances can lead to neurocognitive and other congenital changes to the fetus. Some children begin life with an elevated lead body burden reflecting the lead body burden of the mother.[2] Thus, the development of children born to exposed mothers can be affected, leading to a cycle of susceptibility. The intergenerational effects of child labor may extend to lead exposure if maternal exposure to lead occurs during pregnancy. While not specifically addressed here, this exposure may take place via several routes: mothers working while pregnant, airborne contamination, and contamination of food and water.

Due to these potential intergenerational effects of lead and other toxic substances, child laborers may begin life with lower intelligence or other neurocognitive changes. Thus, when the International Labour Organization's (ILO) Convention 182 injunction against child labor "likely to harm the health . . . of children" is considered in a public health context, the definition of hazardous work becomes quite complex.[6]

Often, children are working illegally or unofficially as part of their family. Children may be working in remote areas and may be impossible to trace through the years.[4] Birthdays may be unknown, and families may not have fixed addresses. In addition, the exact nature of the exposure is usually unclear or hidden. The field of industrial hygiene in developing countries is not well-established and hazard reporting and monitoring capabilities are limited.[7]

This article provides an overview of the extent of hazardous lead exposure in child laborers. Lead was selected because it has been relatively well studied in the general population; however, it is meant to serve as a model for the effects of more numerous, but less studied, substances.

NEUROTOXIC EFFECTS OF LEAD ON CHILDREN

While there are many health effects attributed to lead exposure, this discussion will focus on the neurocognitive changes seen in children, primarily because the link with intelligence quotient (IQ) provides a quantitative basis for discussion. According to the ATSDR, lead is especially toxic to young children and is associated with "decreased intelligence, impaired neurobehavioral development, decreased stature and growth, and impaired hearing."[2]

The development of the brain and nervous system begins in the early stages of embryonic life and continues beyond birth into adolescence. Disruptions of this well-organized series of developmental changes will have lasting effects. Schettler notes that the developing brain is extremely vulnerable to toxic effects, though specific neurodevelopmental data in humans are usually unavailable.[8]

Lead exposure in children has been linked to decreased cognitive function and adverse neurobehavioral effects. A meta-analysis of 24 studies of childhood lead exposures related to IQ concludes that lead impairs children's IQ at blood levels less than 10 μg/dL.[9] Studies have documented lead exposure and decreased cognitive function from age 24 months through age 10 with decreased scores on the Wechsler Intelligence Scale of 5.8 points for each 10 μg/dL increase in blood lead at age 24 months.[10,11]

An 11-year follow-up of subjects who had been studied as primary school children showed that those with higher blood lead levels than their peers had consistently lower IQ scores in fifth grade and subsequently had lower class standing in high school, increased absenteeism, poorer hand-eye coordination, and longer reaction times, among other neurological findings. The authors concluded that exposure to lead in childhood is "associated with deficits in central nervous system functioning that persist into young adulthood."[12]

Despite continued academic discussion, there is widespread acceptance that lead exposure in childhood causes decreased intelligence and that this effect is present even at low lead levels that are still prevalent in the United States.[13] A generally agreed upon numerical estimate suggests that for each increase in blood lead of 4 μg/dL, there is a one-point drop in IQ.[14]

The effects of lead on delinquent behavior and antisocial tendencies are equally significant.[15] Of great concern are the recent findings suggesting that even low level lead exposure can have a significant effect on a child's neuropsychological development.[16] Wigg notes that "lead exposed children are at increased risk of adverse effects on both cognition and learning."[16]

As with intelligence, the neurobehavioral impact of early childhood lead exposure can persist into high school and beyond. This is confirmed by data from the ATSDR showing that young adults who had elevated blood lead levels in childhood had poor scores on neurobehavioral tests 15 to 20 years after their initial exposure.[2] In addition, a health assessment of Idaho residents who lived near a mining and smelting facility demonstrated a dose response relationship, with the severity of dysfunction increasing with increasing bone lead concentrations.[17]

As more data have become available on the adverse health effects of lead, the acceptable blood lead level has decreased. In 1990, the U.S. Department of Health and Human Services aimed to eliminate blood lead levels greater than 25 μg/dL; the new goal in the United States is to eliminate blood lead levels greater than 10 μg/dL by the year 2010.[18] The threshold level for harmful effects of lead is currently unknown.[18]

OCCUPATIONAL EXPOSURE OF CHILDREN TO LEAD

There is almost no information available on toxic occupational exposures in children.[19,20] Data are lacking on all aspects of this subject: numbers of children exposed, duration of exposure, substances involved, resulting illnesses, and long-term effects. Data were unavailable by country, by region, or globally. We attempted to determine the number of industries around the world that use lead and the total number of people potentially exposed to lead at work, but no accurate data were available.

Our exhaustive search for data on lead exposure in child laborers identified only three manuscripts. Others have encountered the same difficulty: "Official data on child labor are generally fragmentary, underestimate informal sector work and family work, and frequently exclude unpaid work."[7]

Children may be exposed to lead in the production of ceramic pottery, battery recycling, stained glass production, automobile radiator repair, construction, and glassworks.

Lead exposure also occurs in the smelting and mining industries and through exposure to fuels.[20] In addition, children's industrial exposures to lead are superimposed on higher environmental exposures seen in developing countries where leaded gasoline is in use.[21]

An observational study of the ceramic tile industry in Victoria, Ecuador, describes the local production of roof tiles.[22] Approximately half of the families in this area are involved in producing roof tiles or ceramic objects, usually at small work sites situated next to their homes. Clay is extracted from the local mountains and then molded into tiles or artifacts. The items are glazed with lead salts made from melted batteries. The ceramics are fired in furnaces for 24 to 48 hours, permeating the local air with dust and fumes from both the ceramics and the fuel. No environmental precautions are in place.

Children as young as 6 years of age were observed working in this trade. Harari et al. enrolled 12 children in a cross-sectional evaluation of lead levels.[22] Ten of these children aged 6 through 15 gave blood samples, and lead levels ranged from 23 to 124 μg/dL, with a mean of 70. Three of the children had worked for less than three months; their blood lead levels were less than 50 μg/dL. All of the children who had worked longer than three months had blood lead levels greater than 60 μg/dL. Corresponding IQ tests were not done, but five of the children had repeated one or more years of school.

Excessive lead levels have been observed in child laborers in occupations where lead exposure is not traditionally suspected. Children who work as scavengers and street vendors also are at risk for hazardous lead exposure. A Philippines study of the child scavengers who worked and lived on Smokey Mountain, a garbage dump that received approximately one-third of Manila's garbage before it closed in the 1990s, found that the 20,000 residents were exposed to waste from chemical, hospital, and slaughterhouse sources.[23]

Children start scavenging when they are as young as 5–7 years old. They begin with a two-year "apprenticeship" and work without any protective equipment. A 1991 survey of 231 scavengers aged 6 through 15 years, recorded mean blood lead levels of 28.4 μg/dL (standard deviation=11.5). Of the boys, 68.2%, and, of the girls, 58.2% had blood lead levels greater than 20 μg/dL. The authors compared these data to blood samples from 25 school children in metropolitan Manila who had a mean blood lead level of 11 μg/dL (Unpublished manuscript. Torres ED, Subida RD, Rabuco LB. Health profile of child scavengers in Smokey Mountain—Balut, Tondo, and Manila. German Agency for Technical Cooperation and International Labour Organization; 1994).

Street vendors also are at risk for lead exposure. A 1993 study of 387 school children and 101 child vendors aged 6 through 14 in Manila demonstrated that 10.3% of the school children had blood lead levels in excess of 20 μg/dL as compared with 32.7% of the street vendors. The mean blood lead level in the street vendors was 17.8 μg/dL (Unpublished manuscript. Martins JM. Philippines environmental health assessment: an agenda for action; October 1995).

Nutrition and child labor

Consideration of the effects of toxic exposure in child workers needs to take into account other pertinent public health, economic, and social factors. For example, nutritional status is an important determinant of the impact of lead during early development. Iron deficiency anemia is perhaps the most critical public health issue related to lead exposure and affects more than 3.5 billion people worldwide.[23]

Iron deficiency anemia and other indicators of poor nutrition are associated with poverty, and child laborers in developing countries are most often from poverty-stricken families.[24] One would expect significant percentages of child laborers to suffer from malnutrition and micronutrient deficiencies. The data, however, are limited and conflicting. In the Philippines study of child scavengers, the anemia rate was 10% compared with the rate of 27.7% in metropolitan Manila (Unpublished manuscript, Torres EB, Subida RD, Rabuco LB. Health profile of child scavengers in Smokey Mountain—Balut, Tondo, and Manila. German Agency for Technical Cooperation and International Labour Organization; 1994). In a study of male child laborers in Jordan, however, 34.1% of the child workers were found to be anemic.[25] Another study of child laborers in Bombay, India, found an anemia rate of 10% in 73 children in the large slum of Dharavi. The authors speculated that the nutritional status of the Dharavi child laborers is better than in "the great numbers of children elsewhere who do not work but who do not eat."[26]

Of particular concern in the study of child laborers exposed to lead is the fact that children appear to be more susceptible to lead toxicity than adults. Children are more vulnerable to absorbed lead than adults because they develop toxicity at lower blood lead levels.[27] In addition, children absorb more lead from the gastrointestinal tract than adults.[28] Adults absorb 10% to 15% of lead ingested with meals; however, children and pregnant women can absorb up to 50%.[5]

Epidemiological studies have shown an association between iron deficiency and lead poisoning. Willows et al. found evidence of this association among infants with very low blood lead levels (mean=1.7 μg/dL).[29] Wright constructed a model predicting that iron-deficient children are more than four times as likely as their iron-replete peers to have blood lead levels greater than 10 μg/dL when tested at a second clinic visit.[30]

Absorption of lead is even higher in children with low dietary iron, zinc, or calcium intake. Iron deficiency may increase duodenal lead absorption. Low calcium intake activates vitamin D to 1,25-dihydroxyvitamin D, causing increased absorption of calcium and lead in intestinal cells.[5] Low zinc intake could be partly responsible for children's increased susceptibility to lead because lead affects zinc-requiring enzymes in the biosynthesis of heme.[2]

Environmental exposures

The issues of children's toxic exposure in the workplace are further compounded by the lack of separation between the job, home, and the external environment. The lack of distinction between these areas is not limited to obvious examples such as (*1*) child scavengers who live on the garbage heap where they scavenge garbage for resale and basic sustenance or (*2*) the risks of leaded gasoline that cause more exposure to lead than any other source in the world.[31]

Studies unrelated to child labor have documented the

risk of toxic exposures to children from contamination in the homes of exposed workers as well as from environmental sources in industrial areas.[32] An example of occupational disease being transmitted to family contacts in the United States is a National Institute of Occupational Safety and Health (NIOSH) case-control study that examined lead exposure in construction workers and found significantly higher surface lead concentrations in construction workers' homes than in those of controls.[33]

A French study of 125 children with lead-exposed parents found that children's blood levels were significantly higher than the reference group and correlated with their parents' blood lead levels and the lead air concentration at their workplaces.[34] Environmental contamination affecting children was demonstrated in a study of children from a Chinese village with lead smelters located near the residential area. High concentrations of lead dust were found in the air and soil, and on local crops and wheat in the farmers' homes. Mean blood lead levels among children were noted to be extremely high.[35]

UNDERSTANDING THE POTENTIAL COSTS OF LEAD EXPOSURE TO CHILDREN

The model developed by Landrigan et al.,[36] which relies on the calculations by Salkever,[37] was used to assess the costs of childhood lead poisoning in the United States. The methods used by Landrigan were generalized to form a crude estimate of the magnitude of the problem of adverse outcomes (potential percent decrease in lifetime earnings) associated with lead exposure in child laborers. Salkever determined that the loss of one IQ point corresponds to an overall reduction in lifetime earnings of 2.4%.[37] Based on the estimate of a 4 µg/dL increase in blood lead levels causing a 1 point IQ drop, and working on the assumption that there is not a threshold blood lead level for cognitive effects, each 1 µg/dL of blood lead concentration is associated with a decrease in IQ of 0.25 points. Estimates were made for the empiric results as described below.

The Table demonstrates the potential IQ decrease in the three cited studies and the subsequent percent decrease in lifetime earnings. The potential IQ decrease was calculated from the following assumptions: that for each blood lead measurement of 1 µg/dL, there is a mean loss of 0.25 IQ points per child and that the loss of 1 IQ point subsequently leads to a 2.39% loss of lifetime earnings.

Limitations of this analysis include the fact that Salkever's calculations may not be transferable to developing countries. Moreover, the impact of lead on IQ may vary based on the age of exposure and the acute or chronic nature of the exposure. Another concern is that the lifetime expected earnings calculations from the Philippines and Ecuador studies come from the average annual income in those countries: salaries the average child laborer would not be able to attain for socioeconomic reasons before lead exposure and IQ is taken into account. In addition, simulated earnings models are noted to be difficult to compute reliably and accurately.[38]

DISCUSSION

This article documents the potential magnitude of the problem of lead exposure in child laborers. The definition of hazardous employment will need to be considered for jobs with potentially toxic occupational exposures. Low levels of many toxicants will affect early brain growth and development. Substances such as mercury readily cross the placental barrier or are rapidly absorbed from the intestines of small children.[39]

The potential magnitude of the hazard of lead exposure in child laborers is unclear. The remarkable dearth of information adds to the severity of the problem and provides a dramatic example of how toxic occupational exposures in children are overlooked. The definition of hazardous child labor needs to include the consideration of toxic exposures such as lead and other heavy metals.

In addition, the potential for these exposures to interact with pre-existing morbidity such as micronutrient deficiencies should be considered.[40,41] One example of such an effect is the likely combined impact of lead and iron deficiency on brain growth and development. Both iron deficiency and elevated levels of lead have been shown to

Table. Estimated earnings lost due to lead exposure in child laborers

Child laborers	*Source*	*Mean blood lead in µg/dL*	*Potential loss of IQ*	*Percent decrease in lifetime earnings*
Ecuador: ceramic workers	Harari and Cullen, 1995[a]	70.2	17.6	42.1
Philippines: scavengers	Torres, 1994[b]	28.4	7.1	17.0
Philippines: street vendors	Martins, 1995[c]	17.8	4.45	10.6

[a]Harari R, Cullen M. Childhood lead intoxication associated with manufacture of roof tiles and ceramics in the Ecuadorian Andes. Arch Environ Health 1995;50:393.

[b]Unpublished manuscript. Torres ED, Subida RD, Rabuco LB. Health profile of child scavengers in Smokey Mountain—Balut, Tondo, and Manila. German Agency for Technical Cooperation and International Labour Organization; 1994.

[c]Unpublished manuscript. Martins JM. Philippines environmental health assessment: an agenda for action; October 1995.

impair neurological development in young children. Because these conditions may be present at the same time, studies focusing on the effect of one should control for the effects of the second. Failure to do so allows for the possibility that, for example, the effect of iron deficiency on cognitive abilities is confounded by unmeasured lead poisoning.

Lead was used as an example of a child labor hazard in part because of its prevalence and in part because its link with IQ provides a quantitative element that has not been sufficiently studied for other heavy metal and toxic exposures. Despite the many limitations, several conclusions are quite clear. First, information and research on lead and other toxic occupational exposures in child laborers is in its infancy. Second, the magnitude of the problem is immense. Third, additional data will facilitate the capacity of policy makers to determine the types of work that are most detrimental to growth and development. Finally, the potential impact of toxic exposures may manifest in ways that are not traditionally considered by policy makers.

Suggestions that protective equipment might lessen the impact of hazardous exposures are apocryphal. The cost of such equipment is likely to remain prohibitive to some of the world's poorest children and their families. Even a few cents per day may be greater than the cost that can be absorbed by families that must scavenge garbage for their daily food. Also, it is doubtful that protective equipment that has been designed for adults would serve the needs of children, even if it were available. Lastly, some protective equipment is difficult to use or requires maintenance programs. Training is unlikely to take place, and maintenance programs are expensive.

Efforts to improve nutritional status and micronutrient replacement are crucial. More specifically, a clear policy directive on iron supplementation is necessary because of the relationship between lead absorption and iron deficiency. Unambiguous distinctions between home and work are recommended to prevent adults from bringing contaminants into the home and to ensure that infants and children are not cared for in locations that are contaminated with toxic materials. Global environmental policies such as banning leaded gasoline can also find support from this perspective. Finally, this overview of lead exposure in child laborers provides support for the implementation of ILO Convention 182 in developing countries.[6]

REFERENCES

1. Fassa AG. Health benefits of eliminating child labour. Geneva: International Labour Office; 2003.
2. Amler RW, Smith L, editors. Achievements in children's environmental health. Atlanta: ATSDR; 2001.
3. International Labour Organization. Federal Democratic Republic of Ethiopia: child labor survey report—2001 [cited 2005 July 4]. Available from: URL: www.ilo .org/public/english/standards/ipec/simpoc/ethiopia/report/et _2001.pdf
4. Parker DL, Bachman S. Economic exploitation and the health of children: towards a rights-oriented public health approach. Health Hum Rights 2001;5(2):93-118.
5. Wigle DT. Child health and the environment. New York: Oxford University Press; 2003.
6. International Labour Organization. Convention 182: Worst forms of child labour. Convention, 1999 [cited 20 June 2005]. Available from: URL: http://www.ilo.org/public/english/standards/ipec/ratification/convention/text.htm
7. Heymann J, editor. Global inequalities at work: work's impact on the health of individuals, families, and societies. New York: Oxford University Press; 2003.
8. Schettler T. Toxic threats to neurologic development of children. Environ Health Perspect 2001;109(Suppl 6):813-6.
9. Needleman HL, Gatsonis CA. Low-level lead exposure and the IQ of children. A meta-analysis of modern studies. JAMA 1990;263:673-8.
10. Bellinger DC, Stiles KM, Needleman HL. Low-level lead exposure, intelligence, and academic achievement: a long-term follow-up study. Pediatrics 1992;90:855-61.
11. Bellinger DC, Sloman J, Leviton A, Rabinowitz M, Needleman HL, Waternaux C. Low-level lead exposure and children's cognitive functions in the preschool years. Pediatrics 1991;87:219-27.
12. Needleman HL, Schell A, Bellinger DC, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. N Engl J Med 1990;322:83-8.
13. Bellinger DC, Needleman HL. Intellectual impairment and blood lead levels. N Engl J Med 2003;349:500-2.
14. Wakefield J. The lead effect. Environ Health Perspect 2002;110:A574-80.
15. Needleman HL, Riess JA, Tobin MJ, Biesecker GE, Greenhouse JB. Bone lead levels and delinquent behavior. JAMA 1996;275:363-9.
16. Wigg NR. Low-level lead exposure and children. J Paediatr Child Health 2001;37:423-5.
17. Williamson GD, Lybarger JA, Millete D, Berger-Frank S. Lasting neurological impairment from lead exposure in childhood (a cohort study of current and previous residents of the silver valley: assessment of lead exposure and health outcomes). In: Amler RW, Smith L, editors. Achievements in children's environmental health. Atlanta: ATSDR; 2003. p. 54-5.
18. Meyer PA, Pivetz T, Dignam TA, Homa, DM, Schoonover J, Brody D. Surveillance for elevated blood lead levels among children—United States, 1997–2001. MMWR Surveill Summ 2003;52(10):1-24.
19. Landrigan PJ, McCammon JB. Child labor: still with us after all these years. Public Health Rep 1997;112:466-73.
20. Woolf AD. Health hazards for children at work. Clinical Toxicol 2002;40:477-82.
21. Donohoe, MT. Correspondence, "Given the journal's . . .". N Engl J Med 2003;349:501.
22. Harari R, Cullen M. Childhood lead intoxication associated with manufacture of roof tiles and ceramics in the Ecuadorian Andes. Arch Environ Health 1995;50:393.
23. World Health Organization. Life in the 21st century: a vision for all. Geneva: WHO; 1998.
24. Wadsworth M. Early life. In: Marmot M, Wilkinson RG, editors. Social determinants of health. Oxford: Oxford University Press; 1999. p.46-7.
25. Hawamdeh H, Spencer N. Work, family socioeconomic status, and growth among working boys in Jordan. Arch Dis Child 2001;84:311-4.
26. Mehta MN, Prabhu SV, Mistry HN. Child labor in Bombay. Child Abuse Negl 1985;9:107-11.
27. ATSDR. Toxicological profile for lead (update) [cited 2005 July 4]. Available from: URL: http://www.atsdr.cdc.gov/toxprofiles/tp13.html
28. Timbrell J. Introduction to toxicology. New York: Taylor & Francis; 2002.
29. Willows ND, Gray-Donald K. Blood lead concentrations and iron deficiency in Canadian aboriginal infants. Sci Total Environ 2002;289:255-60.
30. Wright RO, Tsaih SW, Schwartz J, Wright RJ, Hu H. Association between iron deficiency and blood lead level in a longitudinal analysis of children followed in an urban primary care clinic. J Pediatr 2003;142:9-14.
31. Landrigan PJ. The worldwide problem of lead in petrol. Bull World Health Organ 2002;80:768.
32. Knishkowy B, Baker EL. 1986. Transmission of occupational disease to family contacts. Am J Ind Med 1986;9:543-50.
33. Piacitelli GM, Whelan EZ, Sieber WK, Gerwel B. Elevated lead contamination in homes of construction workers. Am Ind Hyg Assoc J 1997;58:447-54.
34. Laforest L, Annino MC, Alluard A, Precausta D, van den Wiele F, Albouy J, Jehanno F. Epidemiologic study of lead contamination of

children of occupationally exposed parents. Rev Epidemiol Sante Publique 1999;47:433-41.
35. Wu Y, Huang Q, Zhou X, Hu G, Wang Z, Li H, et al. Study on the effects of lead from small industry of battery recycling on environment and children's health. Zhonghua Liu Xing Bing Xue Za Zhi 2002:23;167-71.
36. Landrigan PJ, Schechter, CB, Lipton JM, Fahs MC, Schwartz J. Environmental pollutants and disease in American children: estimates of morbidity, mortality, and costs for lead poisoning, asthma, cancer, and developmental disabilities. Environ Health Perspect 2002;11:721-8.
37. Salkever DS. Updated estimates of earnings benefits from reduced exposure of children to environmental lead. Environ Res 1995;70:1-6.
38. Holmer MR. Validating simulated earnings histories. U.S. Department of Labor and U.S. Social Security Administration; 2000 [cited 2005 July 4]. Available from: URL: www.polsim.com/ehvalid.pdf
39. Wallinga D. Preventing pollution protects children. Healthy Generations 2004;4:8-9.
40. Mahaffey KR, Annest JL. Association of erythrocyte protoporphyrin with blood lead level and iron status in the Second National Health and Nutrition Examination Survey, 1976–1980. Environ Res 1986;41:327-38.
41. Clark M, Royal J, Seeler R. Interaction of iron deficiency and lead and the hematologic findings in children with severe lead poisoning. Pediatrics 1988;81:247-54.

Photo: David L. Parker

Looking for conch shells in the mangrove swamps of Nicaragua (2004)